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A NEW SCOTOMETER.

By COL. R. H. ELLIOT, I.M.S.

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THE BRITISH JOURNAL
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FEBRUARY, 1921

COMMUNICATIONS

SNOW-BLINDNESS:

Its causes, effects, changes, prevention, and treatment*

BY
SURGEON-COMMANDER E. L. ATKINSON, D.S.O., A.M., R.N.,
R.N. COLLEGE, GREENWICH.

THESE observations on snow-blindness are from actual experience of this affection, first in the Antarctic in 1911-13, later, and, to a less extent, in France during the winter of 1916-17, and again in Russia, on the Northern Front, in 1919.

The following investigations are due to experiments made during these periods.

It would be as well to contrast with this affection the results on other workers whose occupation necessitates their dealing with strong lights, whether sunlight or artificial, and whether the excessive exposure is sudden or prolonged. The two conditions used for contrast are in telescopic observations of the sun, and in welding operations with high power electricity. In the former, there is prolonged exposure to intense illumination, and in the latter, a blinding flash. In either case, the effect occurs only with the unprotected or insufficiently protected eye. In telescopic blindness the complaint is of an immediate positive central scotoma, and this may be absolute. The colour vision is affected centrally, and this may extend peripherally. There is an oscillatory

* Paper read before War Section, Royal Society of Medicine, on December 13, 1920

movement and metamorphopsia, with usually a loss of light reflex at the macula and some pigmentation at the fovea. With the loss of a quarter of the vision complete recovery is seldom obtained.

Electric Ophthalmia.—This comes on within twenty-four hours after exposure to excessive light.

There is conjunctivitis and oedema of the lids, the pupils are contracted, and later there is a muco-purulent secretion. Recovery is complete.

Blinding of the Retina.—The effects may be due to excessive stimulus from the rays; a coagulation of tissue elements is caused, probably due to the heating effect of the red rays.

Snow-blindness.—On the Barrier the conditions causing snow-blindness vary, and give rise to different symptoms.

(*a*) The effects in the first class of case to be described are due to exposure to excessively strong illumination by the sun at certain maximum altitudes. There is a selective capacity also for the ultra-violet and violet end of the spectrum, by means of reflection and refraction of the light, from and through the surface of ice-crystals. On such days the actinometer time is as low as 1.25 seconds, in California the lowest recorded time by this same actinometer is one second. On these days exposure of the unprotected eye for 15 minutes was sufficient to bring on an attack. The first symptoms occurred on entering the tent, when there was immediately a feeling as of grit in the eye, with lacrymation, photophobia, chemosis and, if prolonged exposure had taken place, corneal ulcers. There was a hyperaemia of the retina and blurring of colour vision. With treatment the condition lasted 48 hours and recovery was complete.

(*b*) In the second form the effects are partly due to selective illumination, but in addition, a diplopia lasting several days occurs. This is probably due to tiring of the internal and external ocular muscles, and is caused by difficulties of vision through lack of contrast. Lacrymation, photophobia, and chemosis are less marked and there are no retinal changes. The diplopia persists for several days, but recovery is complete. On the calm overcast days giving rise to these latter conditions, the strength of light, measured by actinometer, varies from 2 to 12. The atmosphere is filled with countless numbers of ice-crystals falling slowly downwards with a side to side dipping movement. They cause such diffusion of light that often one can be standing alongside a 9 feet cairn built of slabs of snow, and visually one cannot appreciate the fact, but only by touch. On such days the sun may be invisible. The horizon, barrier surface, and sky are blended by the greyish white colour, and the impression is that of progress within a globe of this solid colour.

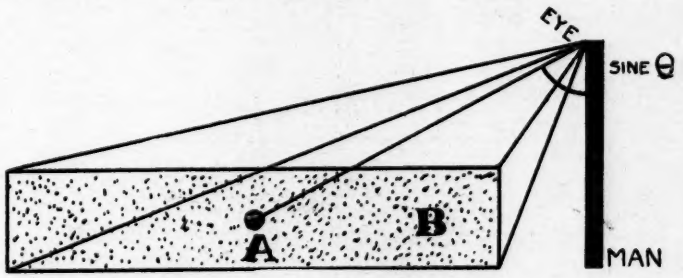
The conditions described above will show clearly that on these

days the effects caused cannot be attributed to direct action from glare. There is, however, the same selective capacity of the ice-crystals for the violet end of the spectrum, and this furnishes further presumptive evidence that these rays are the cause of snow-blindness.

Efficient protection against either condition is given only by glasses such as red and amber glasses which cut out definitely the violet and ultra-violet end of the spectrum.

Beyond the slight hyperaemia occurring in the first form there are no retinal changes; this hyperaemia may also be reflex. The onset of the initial symptoms varies in period of time. It has been stated that the ultra-violet ray is incapable of passing the lens. The violet ray, however, can pass the lens and impinge upon the retina. The intensifying of this ray under the conditions described would cause snow-blindness. In addition, the actual number of the rays of light entering the eye is increased with the maximum altitude of the sun, and a part of the affection may be due reflexly again to the intensity of illumination from any wave-lengths impinging upon the retina.

The following diagram will serve to show the cause of this increase of intensity of illumination under the circumstances.



Contrast between image of Sun A on a perfect surface and B a perfect distributing surface.

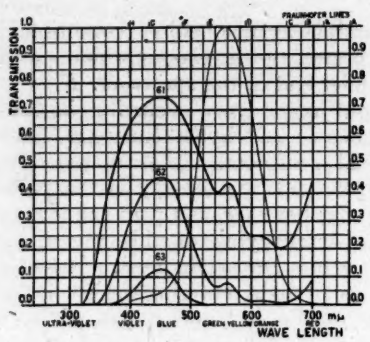
B Represents a collecting field of vision composed of barrier surface, the rays of light reflected from this being collected at the eye.

If B were a perfect reflecting surface the sine of the angle θ does not change.

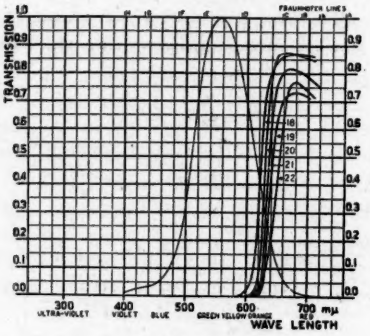
Given, however, a perfect distributing surface such as is formed by the felted masses of ice-crystals, forming the barrier surface, the number of rays collected at the eye will increase directly with the increase of the altitude of the sun.

This was proved by experience to be the case. Travelling across the barrier was done at night, the coldest time, so that the animals

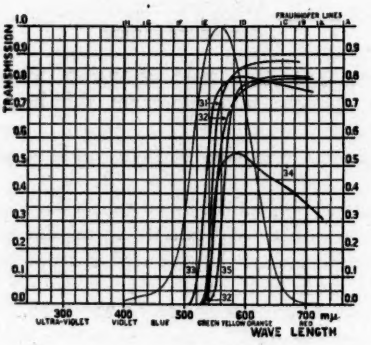
could rest during the warmer day. During these times the sun was at its lowest altitude, no glasses were worn, and there were no cases of snow-blindness.



Blue glasses.

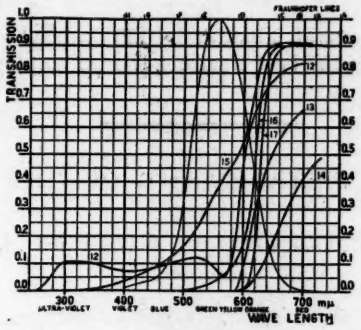


Red glasses.



Orange yellow glasses.

On reaching the foot of the Beardmore Glacier the routine was changed to day marching. No glasses were worn, and by night 50 per cent. of the party were snow-blind. It is presumed that the causation was mainly due to the effect of the violet end of the spectrum. Glasses giving best protection always occlude this end;

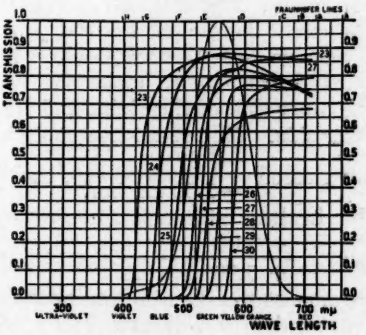


Variation in samples of red glasses

with light blue glasses, which do not do so, snow-blindness can occur.

There is a well-marked selective capacity of the ice-crystals for this ray which intensifies its effect.

In the second form, attributed partly to muscle tiring through lack of contrast, the efficient use of glasses also serves to lessen the strain.



Orange yellow glasses.

Red and amber glasses will give the most efficient contrast on these dull days, and enable objects to be picked up more easily.

The tables serve to show the results of transmission of rays by numerous samples of glass. The glasses used were compared with German samples obtained before the War.

The variation between some of the glasses will serve to show the importance of testing each sample before use with at least a comparative spectroscope. Efficient goggles are made of soft leather, and it is important that side protection should be ensured.

If possible the glass should be as far away as possible from the face to prevent condensed vapour being frozen on it in low temperatures.

Hemisine or adrenalin chloride 1:1000 dropped into the affected eye was found to be the best treatment so far employed.

IDIOPATHIC DETACHMENT OF THE RETINA*

BY

CHARLES KILLICK, M.D., F.R.C.S.

BRADFORD.

IDIOPATHIC detachment of the retina as distinguished from that definitely due to trauma, new growth, or general disease is a *bête noire* of the ophthalmic surgeon and for this reason our thoughts should be constantly directed to the subject, especially in view of any new inquiry which may throw light upon it. Accordingly, in this paper I have largely followed Dr. Gonin, of Lausanne, in his recent full and careful description of the pathology and pathological anatomy of the disease.

We may take as a more or less typical case the following:

"Mr. W., age 41, clerk, first consulted me on October 27, 1920, complaining of slight failure of sight in the right eye. On examination a few vitreous opacities were noted, and it was only after careful search that I was able to make out two detachments, one above and to the inner side of the globe, and the other, smaller, below and to the outer side. No retinal tear could be seen. Vision of the right eye with glasses, $\frac{-6.00}{-2.00 \text{ ax. hor.}}$, was 6/24. The left eye appeared to be healthy although of only moderate visual acuity, 6/24 with glasses, $\frac{-3.00}{+ 5.00 \text{ ax. vert.}}$. I recommended complete rest and made arrangements to take him into hospital the following day. The very next morning he returned with a huge detachment in the superior region and his vision was reduced to counting fingers. Treatment consisted of complete rest with atropin drops and pressure bandage; the eye was examined ophthalmoscopically every second day. Firm pressure was not tolerated

* Read before the North of England Ophthalmological Society, Bradford, December 8, 1920.

and a corneal ulcer developed which speedily disappeared, however, on substituting a light pad. After a few days it was noticed that the retina had gone back and after a fortnight he was allowed up with 6/36 vision. Relapse occurred in a few days and he again returned to bed, with the result that the retina once more resumed its normal position. I now cauterized the sclera as far back and as near to the site of the detachment as possible. For reasons of health, he was again permitted to get up after a week's rest, but in spite of taking things very easily, relapse occurred in a day or two; vision sank to counting fingers, and when last seen he had an extensive detachment in the old position. The optic disc was clear, myopic fundus changes were not marked, and the media were not particularly hazy."

Diagrams from Gonin's "Rapport" in *Bull. et Mém. de la Soc. française d'Ophtal.*, 1920.

FIG. 1.

Shows detachment of solid part of vitreous and its separation into stroma and liquid.

It is for this type of case, and for this only, that the term "idiopathic," in default of a better term, should be reserved, though even here there may have been a predisposing cause in some slight injury, remote in time, and then the detachment precipitated by some slight shock or strain, and it may therefore, strictly speaking, be really traumatic.

The chief clinical characteristic in such cases as the above is the fact that it is the superior part of the eye which is nearly always first affected, and central vision may speedily become gravely affected by involvement of the macula. Indeed, we have all seen cases where the disc has been partly or even wholly obscured by a

fold of the retina falling in front of it. In another type of case, however, it is the inferior part of the globe which is first affected. The following two cases may be quoted in this connection:

"Miss S., age 48, consulted Dr. Little in 1918. She had previously been under Dr. Bronner's care, who had kept her in bed for two months and performed an operation upon the eye of the nature of which she was ignorant. Dr. Little's notes state: "Left eye blind from cataract and bad projection, right eye detachment over the lower half of the field, error of refraction $\frac{-5.00}{-3.00 \text{ ax. hor.}}$." No note of the vision was unfortunately made at that time, and no special treatment was given. I first saw her on August 25, 1920, and noted vision 3/60; a retinal tear can be seen to the lower and

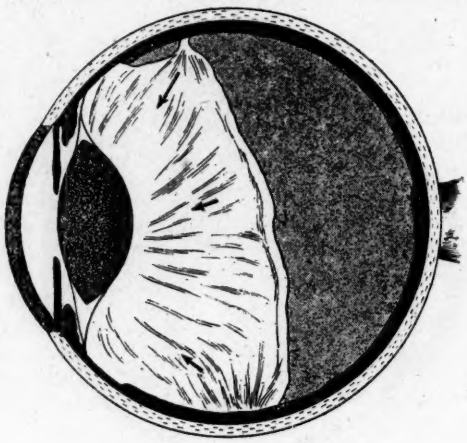


FIG. 2.

Shows adhesion of vitreous to the retina above.

outer side, and the retina is extensively detached in the inferior region. She stated that she was not much worse than she was two years previously, and was able to get along fairly well.

Here then is a detachment in the inferior region which involves the macula, it is true, but which is compatible with a fairly useful amount of sight.

The second case is that of "J.M., age 25, who came to the hospital on October 23, 1920, the history being that he could see black lines in front of the left eye. Vision of the right eye 6/6, left with + 2.00 6/18. Some fine vitreous opacities noticed. On examination, the left retina is detached well forward below, and somewhat to the inner side, grey in colour, no tear to be seen. The lesion

appears to be stationary, and no treatment was considered necessary. Two years previously the patient had attended hospital for a foreign body on the left cornea, which was removed. As the vision was taken, and was the same as at present, it seems likely that the detachment already existed, but there is no note on the case paper of the fundus having been examined."

Here is another example, then, of a detachment existing for a long time quite compatible with good vision, but in this case there may possibly be a traumatic element.

Probably the most important factors in actually bringing about the disaster are primarily an adhesion of vitreous to retina, and secondarily a perforation of the latter due to traction. Gonin's analysis of his own material shows that retinal tears occur in four-fifths of

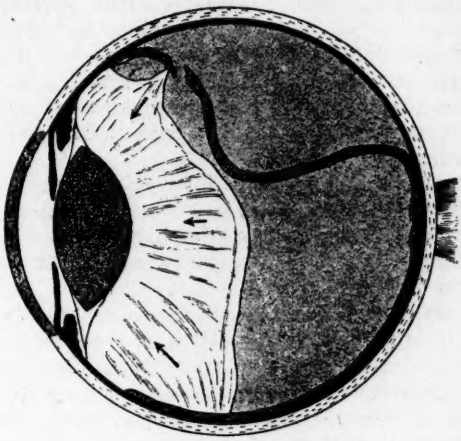


FIG. 3.

Shows the consequences of such adhesion.

the recent cases. He describes three varieties. The majority are valve-like in form, shaped like a circumflex or crescent, with apex or convex border directed towards the disc, and base towards the periphery. Now and then a piece may actually be punched out of the retina, and be seen floating in the vitreous. The third kind is found at the ora serrata, and is therefore difficult to see with the ophthalmoscope. The last-named tend to be multiple, and to form a sort of festoon arrangement.

Whatever the type of rupture it is here in all probability that the detachment has started. As a general rule rapid development in the superior region is associated with a perforation of the valve-like or punched out type; a more chronic onset, on the other hand, involves the lower part of the eye, and if a perforation is present it will be

found to be of the festoon type. Small patches of old choroiditis may often be made out in the neighbourhood of the tears.

The attraction theory enunciated by Leber and supported by Gonin in his recent work assumes that a change occurs in the character of the vitreous. This change consists of a shrinking of the stroma and extrusion of its fluid contents. The stroma or pulp has a greater density than the fluid and tends to gravitate downwards (Fig. 1). If, then, there is a retino-vitreous adhesion secondary to a patch of choroido-retinitis it will be influenced by this change of density according to its situation above or below (*Fig. 2*). If placed in the superior position the weight of the stroma and the movements of the eyeball will exert a traction on the adhesion and will tend to produce a detachment by first causing a tear (Fig. 3). (I take the liberty of reproducing Gonin's diagrams which schematically show how this occurs). In the inferior region, however, there is a somewhat different state of things; here the relations of vitreous and retina will, on account of the force of gravity, be much more intimate. Close contact will promote extensive adhesion and if the membrane becomes detached it will tend to be raised as a whole rather than a tear be produced.

In the rare cases where the superior portion becomes re-attached it must be assumed that the adhesions break or slacken owing to the retina being raised below. On the other hand, a primary detachment below would tend to remain stationary and not to extend, as my second and third cases prove.

The examination of the material at Gonin's disposal demonstrates the fact that the pre- and post-retinal fluids are identical and derived from the vitreous, except in very late stages where glaucomatous changes have supervened, in which case the subretinal fluid comes from an entirely different source, namely from the choroid, and is coagulated by the fixing agent.

The degenerative changes in the vitreous must be attributed to defective nutrition of the uvea, more especially of the ciliary body which undergoes atrophy with proliferation of its epithelium. The epithelial cells wander through the fissures in the vitreous and form bands which still further strengthen the existing adhesions. As atrophy of the ciliary body is not uncommon in myopia, we have here an explanation of the important part which this defect plays in idiopathic detachment. Short sight is not, however, the only factor, though probably the most important one. Gonin's 80 cases were made up of 18 high myopes, 19 moderate (6-10 D.), 16 slight (1-5 D.), 20 emmetropes and 7 hypermetropes. Four patients without any refractive error had double detachment. Again out of the 85 cases of authenticated cure collected by the Ophthalmological Society in 1915-16, fifty-one were myopes.

So long as the vitreous body is healthy, detachment of the

"idiopathic" variety does not occur. In those cases of traumatic choroido-retinitis proliferans with which we are all so painfully familiar, exhibiting, as many of them do, the most profound changes in retina and choroid, detachment, as Wallace has shown, only happens with extreme rarity, twice only in his series, one of which went back after a fortnight's rest. Again we have all met with escape of vitreous after cataract extraction, especially in our less experienced days, but we know that it seldom leads to an untoward result in spite of the fact that the vitreous body is incapable of regeneration; the usual result after a large loss is expulsive haemorrhage. There can be no doubt then that so long as the vitreous is healthy retinal detachment is extremely rare, and the cases I have quoted as occurring in my own practice seem to indicate that Leber's theory has a certain degree of probability, and if so, it must follow that the chance of a spontaneous cure, or of one brought about by remedial measures, must be very meagre, more particularly if the lesion commences in the superior region.

If, however, detachment, has been brought about by choroidal effusion as in cases of trauma, albuminuria, and other diseases, the prognosis need not be so guarded, as the following case shows:

Mrs. T., age 60, admitted to hospital on October 2, 1920, with subacute glaucoma of both eyes, vision of right eye 6/36 and left eye nil. Corneo-scleral trephining was performed on both eyes two days later. On October 19, the notes record—Fundus reflex clear except to outer side where retina appears to be detached far forward and bulges inward, vision 6/60. Left eye retina detached to outer side and below, well forward, media clear.

I saw the above patient again on November 20, 1920: vision, right 6/60, left nil. Every trace of detachment had cleared away in both eyes. I can recall a second patient some years ago, who was under my care at the Kent County Ophthalmic Hospital, who suffered from recurrent attacks of detachment after cataract extraction; he had diabetes, and, I think, albuminuria as well. Rest in bed for a week or two was always successful in bringing about a complete, although temporary, cure.

Consequently it is important that such cases should be carefully discriminated from the idiopathic variety, as the pathology is different, and the statistics of authenticated cure above alluded to are not so valuable as they might be, including as they do 11 of traumatic origin, 6 of albuminuric, one occurring in pregnancy, and two after cataract extraction, out of a total of 85 altogether.

The question, as it seems to me, is largely one of prophylaxis. The improvement that follows careful supervision in the Bradford Myopia School has been recorded by Mortimer in a recent paper, and more will probably be done in this direction in the future. How to maintain the ciliary body and uveal tract in their highest

state of efficiency is a problem that must be solved if we wish to guard any predisposed patient against the awful calamity of idiopathic detachment. With regard to the curative treatment I do not think any rational method has yet been suggested, and we still await the solution of how to restore and keep in proper position a retina which has become detached as a result of secondary changes in the vitreous body.

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EYE DISEASE RESULTING FROM MALNUTRITION

BY

CAPTAIN W. J. RUTHERFURD, M.C., M.D.Glasg.,

LATE R.A.M.C.

AFTER the Armistice I was stationed on the Franco-Belgian frontier, in a district where the civilians had endured continuous hardships and misery under the German régime since the commencement of the war in 1914.

During the final German retreat, they had been forcibly evacuated from their homes by the enemy, being sent off long distances by road or by canal into Belgium and Holland. The winter was coming on, the nights were freezing, the civilians were insufficiently clad and insufficiently nourished, and many of them were debilitated in consequence of the epidemic influenza with which they had been affected. When driven off by the Germans no arrangement seems to have been made to provide the refugees with any food.

After the Armistice they came straggling back to their old homes, returning to a district denuded of food, with the railways destroyed and the canals broken down, and with craters blown in the cross-roads. The feeding of this civilian population was undertaken temporarily by the Army authorities until the French could organize their own *ravitaillement;* and in the First Army area even transport animals were killed, cut up, and distributed as food. In the more rural districts there was a small quantity of root crops and green vegetables remaining, and the people consumed what seed potatoes hey had been able to conceal, for the 1918 harvest and potato crop

had been carried away by the Germans, who had also removed all the live stock.

Naturally, the health of the people suffered as the result of the miseries they endured, and in some of the districts the medical attendance was either inadequate for the time being, or else the supplies of drugs and so on were non-existent, having been removed or destroyed as the Germans withdrew. In consequence it fell to the Army medical officers to do what they could for the sick civilians in addition to carrying out their ordinary duties. This enabled us to see from within, in a way we could never have otherwise done, the sufferings the people had endured; and many an instance of high-hearted courage under tribulation, of devoted patriotism and faith, and of parental self-sacrifice presented itself to my notice while going about among the wonderful French peasantry.

During this time I attended a very large number of civilian cases varying in nature from acute appendicitis to dental extractions, and from multiple shell wounds to uterine displacement. Amongst all these cases requiring attention, there were numbers that could be definitely classified as resulting from the various sufferings and hardships undergone during the war, misery diseases due in the main to malnutrition.

A striking example of this was seen in the case of diseases of the eye. Phlyctenular conjunctivitis, ulceration, and dry conjunctivitis (xerosis) were not merely met with, but were common. Patients came with blepharitis, with hordeola, and with ordinary conjunctivitis, while measles and whooping cough were accompanied by conjunctivitis, ear discharges, and vulvo-vaginal catarrh; and I met with one case each of ruptured dacryocystitis after measles, congenital cystic swelling of the lacrymal sac, and multiple cysts of the tarsal margins; but when these rarities are eliminated, 14 cases were met with in which the eye condition may be said to have depended upon factors such as have just been described. Altogether 1,238 civilian cases of all sorts passed through my hands between the time of the Armistice and the date when I was demobilised, and of these 1.13 per cent. were of this nature, a proportion enormously greater than could ever be met with in a mixed civilian practice under normal conditions.

Treatment of these cases was sometimes difficult. They ought to have had extra nourishment, and especially required extra fat, either as cod-liver oil or in other ways, but this was impossible to procure. The 7th South Staffords, which was for some time in the same district as I was, established a soup kitchen, boiling all the spare bones that could be obtained, and distributing between 30 and 40 gallons of soup every night until the strength of the unit became greatly reduced by demobilisation and the brigade finally moved out of the area; but this could at best make little impression

on a large population. Extra nourishment could be purchased—at a price—across the frontier in Belgium; but in a district where the people were in poverty and all work was at a standstill, it was no easy matter to expend fr. 1.25 for a single egg, or to buy butter at fr.25 the kilo. I procured from a private source a large supply of "Oxo cubes" and distributed them to the eye cases and to other special cases in which impaired nutrition was a factor of importance.

Tonics were administered to all the eye cases under treatment, either in the form of 2-grain quinine pellets, or as the compound tonic pellet containing iron, arsenic, quinine, and strychnine, that could be procured through the Army; but I could never get enough of these, my indents for supplies either being cut down before they were passed on, or not being fully met at the Field Ambulances from which I drew my supplies. Personal interviews with the Commanding Officer of the nearest Field Ambulance, and communications to the D.D.M.S. of the Corps did not result in the supply of anything like the amount of material that could have been made use of, and I carried on from hand to mouth, constantly running out of supplies, but at times succeeding in begging a little from other units, some of which were a considerable distance away. Atropin was used as a local application in all cases of eye ulcers, some of the worst of which were cauterized with liquefied carbolic acid applied either with or without preliminary anaesthesia with cocain. When the atropin ran out and could not be renewed, a small supply of homatropin was made use of. Some cases that seemed to require a nerve sedative were given bromide of potash, and this was thought to be useful. Lotions were made up to bathe the eye at home, either boric, zinc sulphate, permanganate of potash, or a mixed lotion consisting of the permanganate and zinc sulphate together; the water with which the lotion was made being always the chlorinated water taken from the water cart of the unit. It was not advisable to give the patients the solid drugs and allow them to make up their own eye lotions at home, as the local water supplies that I had had tested bacteriologically were found to be grossly polluted, and to contain both free and combined ammonia. But wherever possible the eye cases were personally treated, and I had them coming daily, over a very long period, to the room I used as an Aid Post, the ultimate results in the main being very satisfactory.

The following is a brief *résumé* of the cases referred to:

1. Clementine R., æt. 17 years. 6 December, 1918. Phlyctenular ulcer on left lower sclero-corneal margin. Is convalescent from epidemic influenza, or, as she calls it, "maladie des Boches," and is very debilitated. Suffers from chlorosis and dysmenorrhoea, and has occasional syncopal attacks. 7 December. Phlyctens have developed on sclerotic of right eye as well

10 December. Phlyctens have cleared up entirely from right eye, and left eye is nearly well too. She had several subsequent relapses, however, and was a long time under treatment.

2. Mlle de M., aet. 22 years. 11 December, 1918. Phlyctenular ulcer on outer quadrant of right sclero-corneal junction. Is chlorotic. Was a long time under treatment.

3. Léon R., aet. 8 years. 14 December, 1918. Ulcer on right cornea. Has nebula on left cornea also. Was nearly three months under treatment before he could be said to be cured.

4. Lucienne B., aet. 16 years. 18 December, 1918. Sclerotitis of right eye for past fortnight. 22 December. Has developed several small phlyctens on sclero-corneal margin. Suffers from chlorosis. This case was under treatment for about two months.

5. Justine C., aet. 8 years. 19 December, 1918. Keratitis of right eye for a year, with some slight ulceration, and scleral injection. 21 December. Small phlycten on inner sclero-corneal margin.

6. Mme. M., aet. 41 years. 23 December, 1918. Corneal ulcer on left eye, with pannus of upper corneal segment.

7. Gaston R., aet. 13 years. 7 January, 1919. Bronchitis with dry croupy cough for past four days. T. 102° F. Zone of hyperaemia on inner quadrant of bulbar conjunctiva of right eye, suggestive of an incipient phlyctenular conjunctivitis.

8. Mme. Octave C., aet. 76 years. 21 January, 1919. Keratitis of left eye for past week, with pannus and multiple corneal ulcers.

9. Zélie P., aet. 31 years. 17 February, 1919. Ulcer on upper part of left cornea at sclero-corneal margin, probably originating in a breaking-down phlycten. Condition has been present for a month.

10. Theodule Jules F., aet. 18½ years. 25 February, 1919. Disorganization of left eye by a perforating corneal ulcer (keratomalacia) which ruptured three weeks ago. Dribbles at the mouth and seems semi-imbecile; has a spinal myelitis with thick speech and ataxic gait. This patient was sent to me by a returned civilian doctor with a note requesting me to get him taken into a hospital. The doctor, who knew the family, asserted that patient had a ***myelite specifique***, but I was too pressed with work to make any attempt to verify this. Transport was procured and he was taken into a British military hospital a considerable distance away, with a view to having the disorganized eye removed.

The following are four cases of dry conjunctivitis:

1. Fernand G., aet. 34 years, 27 January, 1919. Xerosis, occurring as a dry conjunctivitis affecting the outer quadrant of the bulbar conjunctiva of left eye.

2. Julienne C., aet. 22 years. 28 January, 1919. Dry conjunctivitis of left eye, with commencing pannus over lower part of cornea.

3. Mme. Sidonie G.-A. 31 January, 1919. Dry conjunctivitis of left eye. Says that she was similarly affected seven years ago.

4. Mme. E.-P., aet. 72 years. 25 February, 1919. Mild chronic conjunctivitis, probably of the xerosis type.

It will be seen that these eye conditions were far from rare among the French civilians who had been subject to the miseries of the German domination for so long; and it is evident that prolonged malnutrition was of great importance as a causative factor, even in cases where the condition was nothing worse than an infection by the *Bacillus xerosis*.

E. C. Bulley has published a "Note on Xerophthalmia in Rats" as the result of certain observations on the consequence of defective diet (*Biochem. Journ.*, Vol. XIII, No. 2, July, 1919), and in an editorial in the *British Medical Journal* for August 23, 1919 (p. 240) the following, with regard to such feeding experiments, occurs and is worthy of note: "In some experiments by Hopkins, the eye condition, which developed within a fortnight after the animals had been put on a diet deficient in the fat soluble factor, was conjunctivitis with ulceration of the cornea, which might go on to rupture of the cornea and dissolution of the globe." Mouquet (*Rev. Méd. Vétérinaire*, Nov., 1919) has recorded the occurrence of nutritional keratitis in five pet dogs whose owners had kept them on a dietary of boiled rice, proved by the event to have been insufficient for their needs. In the profound nutritional disturbances that follow upon systemic poisonings, corneal ulceration may sometimes occur: I have seen it develop in a child who died in secondary coma and acetonaemia fifty-two hours after having swallowed some laudanum that had been left out where he got access to it, and I have also seen a punched out corneal ulcer in a dog dying of a gross overdose of extract of male fern that had been administered by a veterinary surgeon.

HEREDITARY CATARACT

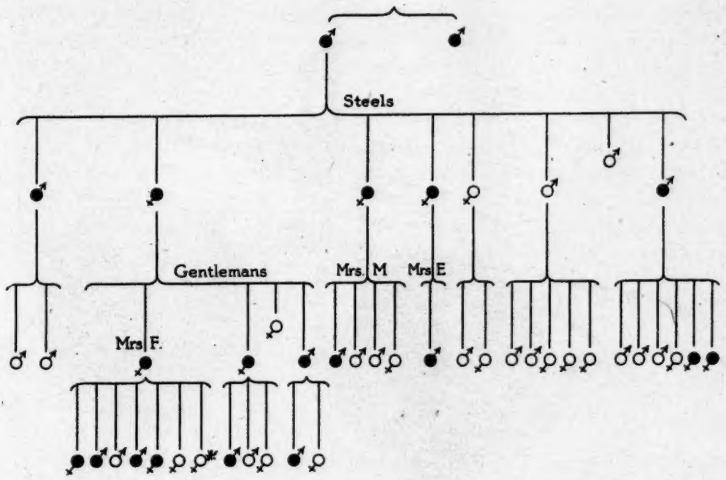
BY

JOHN ROWAN AND JAMES ALEXANDER WILSON

SURGEON, OPHTHALMIC INSTITUTION, GLASGOW ROYAL INFIRMARY. GLASGOW.

SOMEWHAT accidentally we discovered that we had each operated for cataract on different members of the same family, and our mutual investigations have enabled us to construct the following genealogical tree. It is only right to state that we have not seen all these individuals. Mrs. F. has supplied us with most of the information about her relations. All these people, except her

grandfather's brother and one of her cousins, were operated on in Glasgow, several of them by the late Mr. Henry E. Clark. In all the cases the cataract came on between the ages of 13 and 18 years, and previous to this the vision was good. After operation, and with the usual corrections, they seem to have obtained good vision. The cases that we have seen in the early stages presented opaque radiating spokes like ordinary senile cataract. Both eyes are usually affected. Mrs. F. states that her family and her relations are all healthy people.



*NOTE.—Since the above was written it has been found that Mrs F.'s youngest daughter has cataract.—J.R.

A GALILEAN ADAPTER

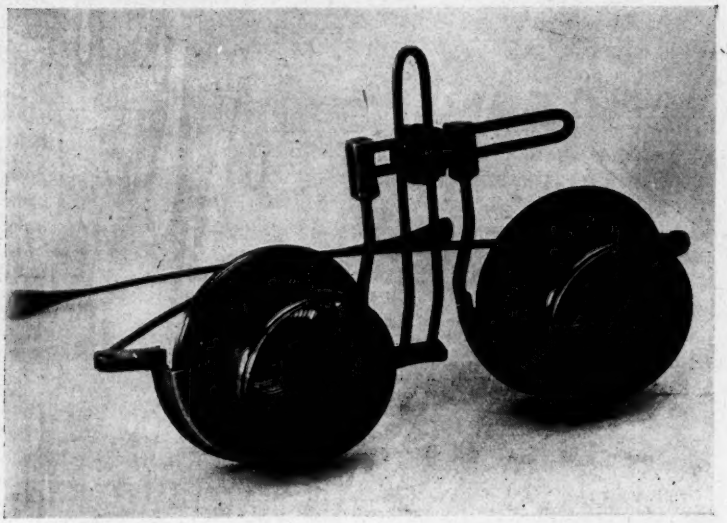
BY

H. GRIMSDALE,

LONDON.

IN The *Ophthalmoscope* for 1911, on page 41 *et seq.*, there is an account of telescopic spectacles which probably everyone who has tested them has found of very varying utility. Some patients rejected them; others found them of great service, and it was not possible to foretell their value in any given case. This uncertainty, together with their considerable cost, was sufficient to make one chary of ordering them, and yet on occasion they gave great help.

In my personal experience, I have found few patients who appreciate the larger image for distance; in most cases, where it is desired to have a large image when reading, it can be obtained by shortening the reading distance, but there are a few people for whom this is very inconvenient; such, for example, are musicians, who must read at a considerable distance, and those whose professions entail much reading aloud, when the face must not be buried in the book. To these the spectacles are often of great assistance.



This adapter, which has been made by Mr. Coppock, of Messrs. Leon and Co., 7, Piccadilly Arcade, allows the surgeon to make a trial so as to gauge its probable value to any patient. It can be placed in the front cell of a trial frame and worn with the patient's static correction. The combination chosen allows an eye made emmetropic, to read at a distance of about 16 inches and gives a magnification of 1.5.

If it be desired to try the effect for distance, a concave lens of about — 4.0 D. must be added to the "distant" correction; the exact strength varies a little with the distance between the adapter and the added lens.

CLINICAL NOTES

INTRA-OCULAR GLIOMA OF OPTIC NERVE OF EACH EYE*

BY

J. LOCKHART GIBSON, M.D.Edin., M.R.C.S.Eng.

BRISBANE.

I FIND that, according to Adami's classification, gliomata belong to the hylomata or pulp tumours, and to the sub-division of pulp tumours he calls epihylomata. Epihylomata are defined "as tumours whose characteristic constituents are overgrowths of tissues derived from the embryonic pulp of epiblastic origin."

Gliomata are the most typical tumours of this sub-division.

All previous intra-ocular gliomata I have myself seen have grown from the retina. When I discovered that each glioma in this case appeared to grow from the optic nerve and not from the retina, I had to call to mind that the optic nerve and retina are both parts of the brain and that a glioma could have its origin from the neuroglia of the optic nerve as well as from that of the retina or of the brain.

The child, aged 7 months, was brought to me with the following history:

Influenza lately, and child kept eyes closed during the attack. Noticed a week ago that child did not see; said to have noticed things until three weeks ago. Six other children all well. The next youngest two years old.

I found slightly shallow anterior chambers, some posterior synechiae: increased intraocular tension (+ 1) and unmistakable glioma reflexes from behind each pupil.

I removed both eyes next day, after explaining to the parents what was the alternative. The child nearly "went out" after he was removed from the operating table. According to reports he improved remarkably in health from the time of operation. There has been no return.

Each eye was put into 2 per cent. formalin at once. In each case the tumour, you will note, is attached by a stalk to the optic nerve. There was no real vitreous, only fluid. The tumour in each case is a large pear-shaped growth stretching from the optic nerve to the lens. In the one eye the retina is still in position everywhere. In the other it has collapsed around the tumour. The section of the tumour confirmed the diagnosis of glioma and proved it to be of highly cellular nature—branching cells with comparatively large nuclei. The nuclei appear to me to be relatively less large than those seen in gliomata of the retina itself.

* Read at the 11th Australasian Medical Congress, Brisbane, August, 1920.

A HIGH DEGREE OF MYOPIA

BY

M. W. B. OLIVER, M.A., M.B., B.C.Camb., F.R.C.S.Eng.

LONDON.

THE patient, J. H., is aged 35 years. He has served for two years in the Army in a labour battalion, and for about one year of this time he was in France.

In the right eye the retinoscopy is — 60 D., and in the left —24 D. His vision in each eye without glasses is less than 1/60 and with the full correction in the right is 3/60, and in the left 6/60.

The fundus in both eyes is normal except that in each eye there is a very small myopic crescent. The absence of any marked fundus changes and the fact that the eyes are not abnormally prominent seems to show that this is probably a congenital condition, the eyes being abnormally large and the orbits correspondingly increased in size.

It has not yet been possible to have an X ray taken of the orbits as the man has considerable difficulty in attending hospital.

AN EYELASH IN THE BULBAR SUBCONJUNCTIVAL TISSUE

BY

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THE following condition being of rather unusual occurrence, appears to be worthy of a brief note.

Private T. was sent on account of defective vision and discomfort in the right eye.

He stated that about five months previously he was injured in the right arm and eye by the explosion of a shell. He was treated in a base hospital, mainly on account of the injury to his arm, the eye condition demanding little attention at the time. Although he did not complain of much amiss with his eye, he has felt, since the injury, that there was something wrong, and he ultimately reported sick, and was sent to this hospital.

On examination the condition was as follows: R.V. 6/12; L.V. 6/6.

The right eye showed little or no sign of lesion on ordinary inspection, but on careful examination with the corneal loupe, a small whitish object was seen at the outer part of the limbus, and was taken to be a "foreign body" lying superficial to the limbus. There was a slight degree of local inflammation involving the cornea

for a short distance. A small round area of greyish colour lay to the outer side of the object, and at a distance of about 3 mm. from the latter.

The eye was well cocainized, and an attempt was made to dislodge the foreign body by means of a blunt spud. It was of minute size, and somewhat mobile, but did not come away at first in front of the spud. After a little manipulation, however, I was able to dislodge it, and draw it downwards along the margin of the sclerotic, when I found, to my surprise, that it drew after it a small hair which had been imbedded in the subconjunctival tissue. The object first observed was obviously the root, which had become slightly bulbous, and opaque from inflammatory changes. The buried end of the hair was white and soft, and had lain apparently close to the greyish region already mentioned.

The eyeball had evidently been forcibly struck by a piece of grit which pierced the conjunctiva at the sclero-corneal margin, and passing in an outward direction carried with it an eyelash, the root of which remained above the surface at the limbus, setting up a slight local irritation.

The fundus was normal, and no further treatment was necessary.

ANNOTATIONS

A Prize in Ophthalmology

THERE can be no greater stimulus to original work than the offering of prizes in special subjects. An excellent example is afforded in this country by the Middlemore Prize awarded triennially by the British Medical Association. Many of the essays written in competition for that prize remain as standard works upon ophthalmological subjects. We have reason, therefore, to be grateful to Mr. W. Edmonds and Miss S. Edmonds, of Wiscombe Park, Colyton, who have founded a prize in ophthalmology in memory of their brother, Nicholas Gifford Edmonds, Lieutenant, 2nd Black Watch, who fell at Magersfontein, on December 11, 1899. The sum of £100 will be awarded every second year for the best essay (which must show evidence of original work) dealing with an ophthalmological theme, and the competition is open to all British subjects holding a medical qualification.

Subject to certain legal regulations, the management of the Gifford Edmonds Prize is vested in the hands of a Committee biennially nominated by the Medical Board of the Royal London Ophthalmic Hospital, who will select the subject of the essay and elect two

examiners. The winner of the prize will be at liberty to give a lecture on the subject at the Royal London Ophthalmic Hospital.

It may be confidently anticipated that the new prize will furnish a strong stimulus to original work in ophthalmology, and Mr. and Miss Edmonds may rest assured that their wish in founding the prize, that suffering may be alleviated, will be amply justified.

The Need of Ophthalmic Laboratories and Museums

It is fully recognized in general medicine and surgery that the only satisfactory method of teaching is attained when there is a thorough correlation of the pathological with the clinical aspects of disease.

The keen surgeon or physician will always substantiate or correct his diagnosis, when the opportunity offers, by careful post-mortem examination; and the clinical teacher stimulates his students most who exhibits specimens and microscopic slides to his class in order to show the exact changes in the organs and tissues which have given rise to the signs and symptoms.

For such teaching a well stocked and well arranged museum, together with a good series of typical microscopic sections or microphotographs, is essential. Successful ophthalmic teaching needs a similar equipment in order to make the diagnosis, prognosis, and treatment comprehensible.

How bewildering it is to students, nay, often to general practitioners, to understand why, in certain cases of glaucoma, eserin is curative and atropin is fatal, while in others the exact converse holds. Yet the mystery is dissipated, directly the anatomy of glaucoma is understood. Again, how difficult it is to comprehend the differential diagnosis between the various forms of pseudo-glioma till one understands their pathology. Or why glioma should appear at one time as a detached retina with a white background, and at others as nodding white masses in the vitreous, till one recognises the two forms of glioma exophytum and glioma endophytum. Examples could be indefinitely multiplied.

While in the throes of reorganization let us see to it that means are provided for students to be taught their pathology—both macroscopic and microscopic—that in our clinical instructions the pathological counterparts are adequately correlated. We cannot do this without ophthalmic laboratories and ophthalmic museums.

Many of our ophthalmic museums are incomplete and are often in a neglected state; too often we see the glycerine jelly melted in which the eyes have been mounted, and the valuable specimens degenerating. There is not sufficient accommodation for the display

of specimens mounted in the beautiful formalin method devised by Mr. Priestley Smith, which is the best mode we yet know of.

Again, in many cases there is no adequate description of the specimens by which their special features can be recognized.

There can really be no lack of material considering the number of eyes which are removed every year, and it is deplorable that the waste of valuable specimens should continue.

Owing to a peculiarity in human nature, we are apt to be more interested in people's failures than in their successes. How stimulating and arresting our teaching would be, if, more than occasionally, it told of our failures and the reasons for them! If this were done more frequently it would also lead inevitably to a demonstration of how our mistakes might be overcome. For instance, what a wonderful collection might be made of both macroscopic and microscopic specimens of eyes lost after *trephining* or after *cataract* extractions. What an opportunity there might be—to give but a single instance—of working out the reason and possibly the cure for the unsatisfactory and often hopeless condition of glaucoma following dislocation of the lens.

We have few laboratories where these eye specimens can be prepared. We must have more, and make greater use of those already existing, and appoint ophthalmic pathologists who have special knowledge in the preparation, the cutting, and the investigation of eye specimens.

Pathology is, as we all know, one of the avenues of medical progress. A thorough grounding in pathology, both for teachers and students, would also act as an incentive to original work. The English medical profession is blamed, not without some reason, for the lack of original work it produces. If English ophthalmology is to fill the position we consider it should hold—and never has there been such an opportunity as the present—there is no doubt that our output of original work must be increased.

ABSTRACTS

I.—THE RETINAL CIRCULATION

Bailliart, P. (Paris).—The retinal circulation under normal and pathological conditions. Report to the Ophthalmological Society of Paris, November 9, 1919. *Ann. d'Oculistique*, November, 1919.

In this communication **Bailliart** summarizes his previous researches, and at the same time carries them a stage further. He considers that the retinal circulation provides the ideal

conditions for the perfect study of the circulation of the blood within an individual organ, and he emphasizes the importance of realizing the large measure in which the circulation in one organ may vary independently of that in other parts of the system.

The retinal pulse.—If the fundus is examined under comparatively high magnification (as, for instance, with a Gülstrand ophthalmoscope), an arterial pulse can be demonstrated in the retinal arteries even under normal conditions. It is, however, better to reserve the term "retinal pulse" for the appearance of this phenomenon under abnormal conditions, as has hitherto been the custom.

When the intraocular pressure equals the diastolic pressure within the retinal artery, the pulsation witnessed will be at its maximum; this is independent of whether the rise in intraocular pressure is fortuitous (as by finger pressure), or pathological as in glaucoma. A spontaneous retinal arterial pulse is evidence that the intraocular pressure is equal to, or greater than, the local arterial diastolic pressure.

T. Henderson's pressure gauge, shown before the Ophthalmological Society in 1914, is mentioned, and credit is given to him as being the first to use this method of measuring retinal arterial pressure. Bailliart only learnt of this work recently.

Priestley Smith's articles in the BRITISH JOURNAL OF OPHTHALMOLOGY are next discussed, and that writer's estimate that the systolic arterial tension in the retina is between 90 and 100 mm. Hg. is considered as too high. Bailliart mentions von Schultên's observations, quoted by Priestley Smith, and lays stress on the improbability that the retinal and carotid arterial pressures could be so nearly alike as the Finnish observer found them to be.

Next there follows a *résumé* of Bailliart's methods, which have already been so excellently described by Priestley Smith in the BRITISH JOURNAL OF OPHTHALMOLOGY, Vol. II, pp. 487-9, that there is little left to add. Bailliart has modified his estimate of the diastolic and systolic retinal pressures, presumably in deference to Priestley Smith's criticisms as to the normal intraocular pressure; his views on this point have also been altered in the same direction by some fresh experiments he has made on the living cat. His figures are a little lower in the child and a little higher in the aged than they are in the average adult. Fasting materially lowers the retinal arterial tension, which is, on the other hand, quite unaffected by sitting as compared with lying.

Hypertension in the branches of the retinal artery.—Bailliart prefers to express this directly in figures read off on his dynamometer, rather than to reduce it to mm. of Hg., rightly holding that the former is both more accurate and more scientific as a method of stating his case. He would, therefore, say that, if the dynamometer

reading is above 30 grammes for the appearance of arterial pulsation, or above 80 grammes for its disappearance, we may set down the diastolic or systolic pressure as too high.

In subjects with marked hypertension, it is possible to recognize the condition by finger pressure alone, but in all doubtful cases, the dynamometer must be used.

Bailliart attaches little importance to the ophthalmoscopic evidence of increased arterial tension as described by Gunn, Bardsley, and others, but would lay much stress on certain subjective signs, which he regards as characteristic. These are: (1) attacks of complete and transitory blindness, which may affect the region supplied by the trunk of the artery, or one or more of its branches, and which speedily disappear under massage. (2) Muscae volitantes, which come and go, and which appear and disappear, independently of the presence of vitreous opacities, and even when the patient is not looking at a brightly-lit surface. (3) The appearance at the periphery of the visual field of sudden, brilliant sparks of light, which appear to circulate within the eye, and to go out as suddenly as they came. (4) A mistiness of vision which causes objects to be seen as through a haze, though no definite clinical phenomena can be found to account for the trouble; Bailliart has no doubt that these are cases of slight retinal oedema.

The causes of retinal arterial hypertension.—General arterial hypertension stands in the first rank of these. As a broad rule, it may be stated that when the general arterial blood pressure rises, the retinal follows suit, but we have also to take account of the factors which may increase the local vascular tension. Of these the most important is vaso-dilatation of the trunk of the retinal artery, which, while lowering the blood pressure above the point of dilatation, increases it in the capillaries and veins below. It is to be remembered that it is in the arterioles of the type of the central retinal artery, that one finds vasomotor contractility at its maximum. Then again, any obstacle to the return of blood in the region of the central vein, will drive up the local arterial pressure independently of the vagaries of the general blood pressure. In other words, the eye manifests the same kind of changes which we know are going on in all the other organs of the system, and, like any one of them, it may be influenced, from moment to moment, by many factors, one of which is the onset of mental emotion.

The complications of retinal arterial hypertension.—The most dangerous of these is haemorrhage. Although retinal arterial haemorrhage may be rare, we must bear in mind that the arterial hypertension brings about changes in the walls of the blood-vessels throughout the whole retinal system, as a consequence, in an early stage at least, of the capillary and venous hypertension which follows in the train of the rise in arterial pressure.

Aetiology.—A local rise in the retinal arterial blood pressure may be merely an evidence of an increase in the general arterial blood pressure.

The causes of the latter may fall with more powerful incidence on the retinal circulation alone; they include albuminuria, diabetes, and syphilis. Of more strictly localized conditions we must mention optic neuritis and thrombosis of the central vein.

Retinal arterial hypotension.—This is much less frequently met with than hypertension. Bailliart has twice seen it in the course of an examination by his dynamometer: the arterial pressure fell, a spontaneous pulse appeared, and then the patients fainted. It must be confessed that, from the surgeon's point of view, this sequence of events might prove very unpleasant and even damaging. de Wecker had noticed the same phenomenon in the fainting, in the dying, and in the subjects of epilepsy, as well as in those in the algid stage of cholera.

The pressure in the trunk of the central retinal vein.—Bailliart believes that the venous pulse is often mistaken for the arterial. The latter is always abrupt, and almost instantaneous; moreover, it affects a considerable part of the arterial network on the disc; on the other hand, the former is slower and more measured, and is generally limited to a very narrow portion of the length of the vein. The vessel seems to give way for a moment, to chase its contents, first towards the centre and then towards the periphery, and finally slowly to resume its normal calibre.

A rarer form of pulse (seen in 2 per cent. of Bailliart's cases) is brought about, not by the movement of the vein walls, but by that of the mass of blood within the vessels; it is a true piston-movement along the axis of the vein. The mass of blood is first pushed centrally, and then returns suddenly towards the periphery as if the *vis a tergo*, for one moment enfeebled, was yielding before a pressure momentarily superior to its own.

The mechanism of the venous pulse.—The current explanations of this phenomenon are stated, and Bailliart then proceeds to discuss the factors concerned in venous pressure: (1) There is first, the *vis a tergo*, originating in the drive from the heart, handed on through the capillaries, and gradually fading away until, in the jugular vein, it may be a negative quantity during the ventricular systole. (2) At the moment of the auricular systole, which just precedes that of the ventricle, the sudden arrest of the venous blood sharply increases the pressure within the jugular. (3) The respiratory movements of the thorax, too, have their influence on the venous pressure; this is positive during inspiration, and negative during expiration. The result is (1) that the pressure in the retinal veins rises to reach a maximum at the moment of auricular systole (which is synchronous

with the ventricular presystole), and (2) that it is lowest during the ventricular systole (which is synchronous with auricular diastole). But during the ventricular systole, the ocular tension is at its maximum, so that at the same moment the walls of the central vein on the disc are exposed to an increase of pressure on their external surface, and a decrease of pressure from within; it is not then surprising that they give way. Next, the ocular pressure falls during diastole, whilst the venous pressure is simultaneously rising to reach its maximum at the moment of presystole (auricular contraction). We can thus understand why the central vein at first fills gradually, and then suddenly, at the onset of presystole, undergoes for a moment a maximum dilatation. All this explains (1) the slowness of the venous pulse, (2) the contraction of the vein, which lasts throughout the systole, and (3) the slow and progressive dilatation of the vessel, during the whole of the remainder of the cardiac revolution.

If the venous pressure, even at its lowest, is higher than the ocular pressure, the pulse we have been describing cannot make its appearance; the piston pulse may, however, be seen, for, when the pressure in the central end of the vein is, during the short moment of presystole, very distinctly above the peripheral pressure, there will be a momentary arrest, or a reflux of the mass of blood within the vein; we then see the distinct and characteristic piston movement, which is most often in evidence when there is an obstruction to the return of venous blood. Lastly, if the venous pressure is constantly lower than the ocular pressure, the wall of the vein, always held down by a superior pressure on its outer side, can never dilate to its maximum; consequently, the vein will be immobile, or at the most, will show but feeble movements.

The information furnished by the examination of the retinal veins.—Bailliart states that it is on the disc alone that the inspection of the veins or arteries can furnish any information of value, for there the vessels are free from adhesions to the subjacent tissues, and so can contract and expand under variations of pressure, putting aside the piston pulse. Two cases present themselves:

(1) *A spontaneous venous pulse is present.*—Here the venous pressure is fluctuating above and below the intraocular pressure. If we would know how far the maximum venous pressure is above the ocular pressure, all we have to do is to press on the globe until the pulse disappears, the dynamometer reading will furnish the information we require.

(2) *There is no venous pulse.*—If, on increasing the intraocular pressure, one appears, we are dealing with an eye in which the venous pressure is steadily above the intraocular pressure. If, on the other hand, no pulse appears, we may gather that the venous exit pressure is steadily below the intraocular pressure, and that the

circulation is maintained through the flattened wall of the vessel, kept sufficiently open for the purpose by its own rigidity. If we lower the pressure of the globe by compression or massage, or if we raise the venous pressure, by making the subject hold his breath after a strong expiration, the venous pulse will appear.

The retinal venous pressure.—The previous estimates of this are first considered. Bailliart's observations on the presence or absence of the pulse in 66 normal subjects are recalled.. Thirty-seven per cent. showed a spontaneous venous pulse; in 12 per cent. a pulse could be elicited by light pressure; in 30 per cent. no pulse could be observed or elicited by pressure; lastly, in 21 per cent., the venous pulse came and went from time to time.

Obviously, then, the venous pressure, at the exit of the vein, is always close to the intraocular pressure, round which it oscillates, influenced by the phases of the cardiac cycle, and by the systolic and diastolic variations of the intraocular pressure. Bailliart would place the intraocular pressure at around 20 mm. Hg., and the venous exit pressure at from 18 to 22 mm. Hg. He argues that, if Leonard Hill was right in placing the pressure in the torcular Herophili at 10 mm. Hg., it must surely be higher in the cavernous sinus, and still higher in the ophthalmic vein and its branches.

Pathological variations in the venous pressure.—Hypertension is found in the veins just as it is in the arteries, but it is a mistake to suppose that a spontaneous venous pulse is an indication of such hypertension, for it may be met with under conditions both of high and low venous pressure. A well-marked illustration of the latter is its presence, many times observed after embolism of the central artery, when the venous pressure is very low indeed. The view that by compression of the globe we can measure the venous pressure, its minimum being signalled by the appearance of a venous pulse, and its maximum by the disappearance of that pulse, is unsound, for the closure of the venous system quickly establishes a condition of uniform pressure throughout the retinal circulation from the arteries to the veins; this pressure is that of the arteries. We can, however, always prove the presence of venous hypertension and we can sometimes measure it: (1) When a venous pulse is present. Whenever it requires a dynamometer pressure of over 25 grammes to stop the venous pulse in these cases, venous hypertension is present and can be measured like arterial hypertension. (2) When there is no spontaneous venous pulse. We can find the pressure at which the pulse is made to appear and that at which it disappears, but as already shown the observation will be open to error.

Practically, we must limit ourselves to noting the pressure at which the venous pulse is extinguished, if spontaneously present, or

induced if not present. These are the only exact measurements we can make. It is sometimes very easy to recognize venous hypertension. When the maximal venous pressure is higher than the minimal arterial pressure, a venous pulse persists after the arterial pulse makes its appearance; this alternation of the two pulses, of which the venous marks the presystole and the arterial the systole, is characteristic of an obstruction to the return of venous blood; it connotes the existence of a vaso-dilatation of the capillaries and veins.

Venous hypertension is frequently met with: In thrombosis of the central vein, it constitutes from the first a diagnostic sign. It is also often met with in retinal haemorrhages. It is a mistake to suppose that it is always associated with arterial hypertension. In albuminuric retinitis, the arterial tension may be very high, and the venous tension normal. Speaking generally, retinal haemorrhages accompany venous hypertension. In glaucoma, retinal venous hypertension is met with, but not by any means constantly.

As for the causes of venous hypertension, these are (1) some obstruction more or less complete, to venous return, or (2) peripheral vaso-dilatation which throws open the capillaries and veins to the results of arterial pressure. Of venous hypotension we know but little. It is certain that, although the retinal circulation can accommodate itself, thanks to the resistance of the vessel walls, to a pressure slightly below the ocular pressure, this inferiority must not be too great, or the venous wall will close, and all circulation will stop.

The relationship between the ocular pressure and the retinal arterial pressure.—Under artificial pressure the visual field is obliterated from within outward, and complete blindness soon comes on. Just before the latter event, the subject can see a rhythmic pulse in his visual field. The appearance of this phenomenon marks the point at which the ocular pressure surpasses the diastolic arterial pressure. When the systolic level of pressure is reached, vision is extinguished. From what we thus see happen under the application of direct pressure, we can argue what will happen if the intraocular pressure rises due to pathological conditions. Nor must we forget that under the latter circumstances the vessel walls will suffer from the increased pressure to which they are submitted. There must be a relationship between the ocular and the retinal arterial pressures. To understand the problems involved we must first study this relationship under normal conditions, and then pass on to consider them under those which are pathological.

(A) *The normal relationship of the ocular and retinal arterial pressures.* Bailliart assumes the intraocular pressure to be one of 20 mm. Hg., but thinks the exactitude of the estimate to be of less

importance than the study of the difference between the ocular pressure and that within the retinal vessels.

Why should a comparatively slight rise in intraocular pressure unfavourably affect vision? Is it that the retinal cells cannot function satisfactorily, except within well-defined pressure limits? No.! For we see them sometimes bear with impunity considerable falls of pressure. Is it not rather that under these conditions the circulatory supply of the retinal elements is interfered with?

Taking the systolic and diastolic arterial pressures as 70 and 30 mm. Hg. and the venous exit pressure at 20 mm. Hg., what is the probable capillary pressure? This must be lower than the diastolic pressure, otherwise the retinal circulation could only be carried on during part of the cardiac cycle: it must, therefore, be lower than 30 and higher than 20. As an approximation, Bailliart puts it at 25. He discusses Priestley Smith's estimate of 39, which he thinks too high.

Supposing that the intraocular pressure goes above 30, what will happen? The diastolic pulse will appear in the large arteries and blood will only circulate during the systolic period; the thin-walled capillaries, with their low vascular pressure and their feeble resistance to the pressure from outside, will ill maintain the circulation; the more peripheral they are, the more effectually will an increase of intraocular pressure cut them off from nourishing the tissues they supply. The lessons are obvious: Whenever the ocular pressure exceeds the capillary pressure, obstruction to the capillary circulation, and loss of vision follow. The further away from the papilla any area is situate, or the longer the pressure is maintained, the greater will be the damage done.

But we know that pressures well above 30 mm. Hg. may be met with without any visual trouble appearing. Bailliart explains this by stating that when the ocular pressure rises, the arterial pressure tends to rise with it, and so, up to a certain point, physiological equilibrium may be maintained by the preservation of the ratio between these rising pressures. He believes, for instance, that in different positions of the body, a constant relationship is preserved between the ocular pressure and the retinal arterial pressure, and that these fall and rise together according to the variations in the position assumed.

(B) *It is a different matter when physiological limits are overstepped;* the equilibrium is then broken. He discusses in turn the conditions arising therefrom.

(1) *Trouble due to an increase of the intraocular pressure.*—If the ocular pressure rises above 30 mm. Hg., the minimal arterial pressure tends to keep a little above it, but so little that the least pressure on the globe causes arterial pulsation. The systolic pressure does not maintain its relative superiority to the diastolic,

and the two therefore come nearer together. Moreover, the arterial pressure is so little above the intraocular pressure as to make it certain that the latter surpasses the capillary pressure. [Reviewer's note.—Such a condition can obviously be only a temporary one, otherwise either all circulation would stop, or the capillary pressure would have to rise to carry it on.]

Even in the trunk of the central artery, the diastolic pressure may be lower than the ocular pressure; a pulse then appears. Such a spontaneous pulse is rarely seen in glaucoma; but, when seen, it is evidence that the blood is only circulating during a part of the cardiac cycle. Herein lies the key to the signs and symptoms of glaucoma. Bailliart repeats the fallacy that the early limitation of the nasal field is to be explained by the longer course of the temporal vessels. The reviewer has shown the futility of this argument on pages 246 to 248 of his book on "Glaucoma."

Bailliart asks: Is it the raised ocular pressure which defensively brings about a rise in arterial tension, or is it the other way about? He also raises the question of the existence of a vasomotor centre, either peripheral or central, which acts only through the vascular system. We need not follow him at any length in these speculations.

(2) *Trouble due to arterial hypotension.*—If, with the ocular pressure normal, or slightly below normal, the arterial retinal pressure falls sharply, we have the same rupture of equilibrium as in glaucoma. The sharp arterial hypotensions, whether general (as in syncope, cholera, and anaemia), or local (as in quinine amaurosis), bring about the same trouble as does ocular hypertension. In such cases, Graefe, by iridectomy, and Rothmund, by paracentesis, re-established the circulation and brought back normal vision.

Another very interesting group of cases is that in which we find the evidence of glaucoma without the signs of increased tension. Bailliart is sceptical as to the existence of transitory increases of pressure in these cases, and prefers to believe that there is a disturbance of the equilibrium between the ocular pressure and the arterial pressure, brought about not by an exaggeration of the former, but by a diminution of the latter. He quotes Fourrière, Schnabel, and Morax in defence of his position. The glaucomatous cup and the shallow cupping of optic atrophy are both the results of a single process, an ischaemia of the nervous tissue of the retina and papilla. In the one case the ischaemia is the result of the pressure exercised on the arterial circulation from within the globe, in the other it is an outcome of a direct diminution of the arterial supply of the retina and optic nerve. Bailliart has been interested in the study of these cases of optic atrophy with cupping, and has elicited the following facts in connection with them: The retinal arterial

pressure is always above the ocular pressure; the difference between the diastolic pressure and the ocular pressure remains normal; the systolic pressure is very variable, sometimes high, sometimes low, sometimes normal, but there is one constant factor in all the cases, namely, the feebleness of the arterial pulse-beat, or in other words, the low amplitude of the arterial oscillations. This feature is a characteristic of conditions of vasoconstriction, or of the commencement of obliteration of the retinal vessels. What is true of the trunk of the retinal artery must be still more so of the capillaries. We may, therefore, assume that over a large area of the capillary network, the vascular pressure is below the ocular pressure. He adds that if these views are accepted, all the cases of glaucoma, without increase of tension, should be classified as glaucoma. The common factor would be a rupture of the normal relationship between the retinal arterial pressure and the ocular pressure. Such a rupture was first pointed out by Sulzer, as characteristic of an attack of glaucoma (*Ann. d'Oculistique*, February, 1897, p. 81).

Much of this argument is interesting and suggestive, but to the reviewer's mind, a needless confusion has crept into the writings on this subject. Optic atrophy with shallow cupping is a very different thing, to his mind, from simple chronic glaucoma with its attendant atrophy. The cupping in the latter case is identical with that which we meet with in typical glaucoma, and which finds a ready explanation in the increase of pressure within the globe. He has found on numerous occasions that patient perseverance with the tonometer has yielded evidence of increased tension in subjects, which might easily have been taken under a less careful examination as supporting the cupping-without-pressure theory. Bailliart's present thesis, though put in different and attractive language, really goes no further than the contentions of previous writers, that arterial sclerosis, so far from favouring an increase of blood pressure within the eye, has in practice the opposite effect.

(3) *Trouble due to arterial hypertension.*—If the pressure in the retinal artery rises, and especially if this rise is accompanied by vasodilatation with a rise in the venous pressure, the relationship between the retinal vascular pressure and the ocular pressure is once again disturbed, but this time in the opposite sense, for the walls of the vessels will be less well supported than they normally are, and haemorrhages and exudations will therefore be rendered likely.

This condition of high vascular retinal pressure is met with in its most marked degrees in albuminuric retinitis. Now, it is known that this increase of pressure appears in the retinal network of vessels before the lesions are seen, thus showing that it is not an effect of those lesions. This is an important fact to remember: in dealing with an affection characterised by oedema and haemorrhages, one must take careful account of mechanical conditions so important

as this, and one must attribute to the vascular retinal pressure the rôle of at least strongly favouring the production of the lesions.

In most cases the ocular pressure remains normal, despite the great increase in the retinal arterial pressures. On the other hand, we know that glaucoma and haemorrhagic glaucoma are not exceptional complications of albuminuric retinitis. Bailliart believes that though retinal arterial hypertension may appear, and may be maintained without the occurrence of a rise in the ocular pressure, the latter is often produced secondarily. It is as though the parallelism between the two pressures has a tendency to re-establish itself when disturbed.

The treatment of retinal circulatory troubles.—These are very difficult to treat, but the more we know of their mechanism, the easier will our task become. Without belittling the importance of a rise in the general blood pressure, we must bear carefully in mind that it is the local vascular pressure which must guide and govern our therapeutics. Bailliart furnishes examples:

Example 1. For one reason or another we suspect that a patient has high tension in his retinal arteries; we measure his general blood pressure and find it raised, and so we treat him with hypotensor drugs—his arterial blood pressure falls it is true, but only as a result of vasodilatation; in his capillaries and veins the pressure will rise, and he will thus be more than ever exposed to the danger of local haemorrhages.

Example 2. Vision is steadily falling and the optic discs are showing cupping, despite the fact that the ocular tension is apparently normal; there are mists, or even attacks of blindness, which last some hours. Strychnine makes the patient worse instead of better. We have here to do with arterial hypertension, but the finest peripheral retinal vessels are in a state of spasm or of obliteration; strychnine, being a vasoconstrictor, simply increases the trouble. This patient, with a high general blood pressure, needs vasodilator drugs; anything which will open his peripheral circulation will be as good for him as it was bad for the previous patient.

The same principles must be applied to local treatment: miotics are said to be vasoconstrictors, and mydriatics vasodilators. Whether this be true or not, pilocarpin, when steadily instilled into the eye, has the most excellent effects in retinal lesions, accompanied by vascular hypertension; on the other hand, atropin is dangerous in this class of cases.

Despite the fact that we cannot observe the choroidal vessels directly, we must not forget their importance. We have means of studying them, the best of which is by the observation of the pulse-beat, as transmitted through the walls of the globe, and measured by one or another of the instruments used for determining the

tension of an eye. Bailliart hopes much from a further study of this subject.

A very interesting discussion followed the reading of this paper. Duverger and Barré have controlled Bailliart's experiments. They hold that his methods of measurement are open to numerous errors, but think that the following general deductions may be drawn: (1) The systolic arterial pressure in the retina is about 100 mm. Hg., and the diastolic 50 to 60 mm. Hg. (2) A continued increase in the retinal arterial pressure has no influence on the ocular pressure. (3) An increase in the ocular pressure has probably no influence on the retinal arterial pressure. (4) The retinal arterial pressure is practically the same as the pressure in the brachial artery, when taken at the eye level; it seems to depend almost entirely on the general blood pressure.

Velter, too, has adopted Bailliart's technique. He has found the difficulties considerable. His results differ from those of Bailliart, and agree with the findings of Duverger and Barré. The retinal arterial pressure does not appear to be sensibly influenced by a rise in the ocular pressure, but runs parallel with the general blood pressure.

Rochon-Duvigneaud urges that we should let our theories wait on facts and not hurry our building. Every time that he has found high retinal arterial tension it has been accompanied by a high general blood pressure. He does not believe that Bailliart has met with cases with normal general blood pressure together with increased tension of the retinal arteries. Retinal haemorrhages are the results of venous stasis and not of increased arterial tension. He cannot follow Bailliart's argument as to the pressure within the retinal veins, especially as other factors have to be taken into account, including the viscosity of the blood and the state of the vessel walls. The arterial pulse is a very inconstant phenomenon in glaucoma, and in many cases of this disease, the retinal blood circulation is maintained all through the cardiac cycle, despite the rise in chamber pressure. He cannot agree that this is due to a purely local rise in retinal vascular pressure, and prefers the view that it is a consequence of the increase in general blood pressure which is so often met with in the glaucomatous. The reviewer would point out that reliable statistics do not indicate any causal relationship between high general blood pressure and glaucoma. As the peripheral pressure increases (due to venous stasis), the diastolic arterial pressure may likewise rise in the overfilled retinal arteries. In doing so it approaches, but can never pass, the systolic pressure. So long, however, as it remains superior to the ocular pressure, no arterial pulse will appear. We know that in glaucoma a rise in the ocular venous pressure is a constant feature of the congestive cases, and it is to the association of venous obstruction with intraocular

pressure (caused by an obstacle to the outflow of aqueous fluid from the eye) that we must look for the explanation of the absence of an arterial pulse in so many of our glaucoma cases.

The reviewer has ventured to give Bailliart's views at some length, because they appear to him to be an extraordinarily valuable chapter in the literature of glaucoma. A little more care in the arrangement of the subjects discussed, and in avoidance of needless repetition, would, he thinks, have added not a little to the merits of the contribution. He has, to a great extent, abstained from criticism, not because he accepts M. Bailliart's views in their entirety, but because his object has been to present them as shortly as possible, consistent with clarity. He would be doing that writer a grave injustice were he to omit to mention that he makes no claim to finality in the views which he has put forward, and that the modesty with which he has advanced them is only equalled by their originality and thoughtfulness.

R. H. ELLIOT.

II.—OPERATIONS

(1) **Knapp, Arnold (New York).—Report of the removal of a tumour at the apex of the orbit, with preservation of the eyeball, in a case of plexiform neuroma of the eyelid. Microscopical examination.** *Arch. of Ophthal.*, Vol. XLV, No. 5, September, 1916.

(1) **Knapp,** met with a case of neurofibroma at the apex of the orbit. The tumour was probably connected with the nasal branch of the ophthalmic nerve; there was also a plexiform neuroma in the lid. The tumour, which measured 22 mm. by 12 mm., was found to be adherent to the sheath of the optic nerve; this adhesion was separated, a capsule was isolated and opened, and the tumour was removed, with preservation of the eyeball.

The literature of the subject is given, and two figures serve to illustrate the paper.

R. H. ELLIOT.

(2) **Aymard, J. L.—A cartilage prothesis for the eye.** *Lancet*, October 27, 1917.

(2) **Aymard,** of the Queen's Hospital for Facial Injuries, Sidcup, says that since his investigations regarding the vascularity of cartilage, he can, with the utmost confidence, understand how cartilage, transferred, rapidly establishes fresh communications with the blood-vessels in its new vicinity and becomes fixed to the capsule in a recent enucleation or to the surrounding tissues in

older standing cases. He illustrates his method of trephining two hemispheres from the eighth costal cartilage and pegging (with cartilage), or fixing together with catgut, these two hemispheres to make a sphere for introduction within the capsule of Tenon. At present the author only uses the method in primary enucleations where there is no sepsis, but suggests that it is worthy of extensive trial in septic cases. Apparently, the usual procedure is for one surgeon to trephine and shape the cartilage while another excises the eye. Apparently the trephine employed, which by the way is very poorly illustrated, is expected to shape as well as cut out the piece of cartilage.

ERNEST THOMSON.

(3) **Herbert, Lt.-Col. H.—Maximal perchloride irrigation of the conjunctiva in cataract operations.** *Indian Med. Gaz.*, November, 1917.

(3) **Herbert's** paper is directed to prove that there is no divergence of views between the reviewer and himself on the subject of the value of the method of perchloride irrigation of the conjunctiva before a cataract extraction is undertaken. In this he is unquestionably correct, and his contention that "it is up to" any who meet with suppuration in their cataract operations, to eliminate it by the use of this method, clamours for wide recognition. The credit of originating the idea of perchloride irrigation, before major eye operations, is often given to Herbert, but the practice dates much farther back than his contribution to the subject, and appears to be due to Major Bamber, of the I.M.S., who brought the method before the Indian Medical Congress of 1894; possibly, it may go even farther back still. Many other surgeons have used this antiseptic irrigation, and far from least among such we must include the name of Lt.-Col. Henry Smith, of Jullundur and Amritsar fame. Few, however, have taken such a part in elaborating the technique and in popularising the method as Lieut.-Col. Herbert. This adds to the value of his present communication, which will well repay the careful study of all who are engaged in the operative practice of ophthalmology.

R. H. ELLIOT.

(4) **Liegard. — Suture of cornea in cataract operations. Advantages of the parallel tunnel stitch with two needles on one thread.** *Ann. d'Ocul.*, Vol. 155, January, 1918.

(4) In the present article **Liegard** reaffirms his views on the simplicity of substituting a horizontal suture for a vertical one and of doing both stages with one thread armed with two needles.

Standing to the right side for the right eye and using the right hand for the needle holder the surgeon passes the needle into cornea

0.5 mm. from limbus, about 12 o'clock, and parallel to it, bringing the point out again by a slight rotatory movement close to the entrance point, this needle is discarded and the second needle is used in a similar way just over limbus, all the time the fixation forceps, with rather projecting teeth, keeping a firm grip on conjunctiva and episclera at 11 o'clock. The needles are passed in and out in one movement and the loop of the thread is situated next the caruncle. After the extraction is completed, the upper thread is pulled on and the knot made. The left eye is done also from the right side over the patient's chest, the needle being passed from without inwards so that the loop lies over towards the outer canthus. In both cases support is taken from the corresponding cheek.

Liegard claims as advantages the fixation at one spot during the insertion of both sutures, the precise and easy adjustment of the space necessary for the knife, the easy access to the eyes when passing in the sutures from below rather than over the eyebrows, the solid and constant point d'appui of the cheeks.

Firm fixation at 11 o'clock by being directly opposite the point of insertion renders this easy, reduces to a minimum injury to conjunctiva and gives more elegance and rapidity to the operation.

Two sketches of the operation and the suture material are given.

W. C. SOUTER.

(5) **Terrien, F.—Muscular advancement with a complementary suture. (Advancement musculaire avec suture de complément.)** *Arch. d'Ophtal.*, January-February, 1918.

(5) In the operative treatment of strabismus, muscular advancement is steadily replacing the older method of simple tenotomy; in a certain proportion of cases a combination of both procedures is advisable.

The weak point in advancement operations is the difficulty or impossibility of retaining the shortened tendon in its new position. The stitches in the sclera remain undisturbed, but those in the cut-end of the muscle, exerting antero-posterior traction (greater in proportion to the degree of shortening), parallel to the muscle bundles, tend to cut through the soft tissues and allow the advanced tendon to slip back. In order to counteract this slipping, **Terrien** advises and practises the application of an extra suture, which acts as a stay and lessens the traction on the main suture. He states that he has operated by this method in a large number of cases, and has had very satisfactory results.

The complementary stitch is applied in the way now described: but a glance at the two diagrams in Terrien's original article will give a clearer idea than any written description. In operating,

Terrien makes a horizontal incision through the conjunctiva over the rectus tendon, and proceeds in the usual way to the division of the muscle and the insertion of a silk suture through its cut-end and then through the conjunctiva and sclera close to the limbus. Before this is tightened, the extra suture is placed in position. A silk thread, armed with two needles, is used, white if the other stitch is black, to avoid confusion. One needle is passed vertically through the superficial layers of the sclera opposite, but a little below the tendinous attachment of the rectus; it picks up about 2 mm. of the sclera, then emerges, and is again inserted for a like distance and the silk pulled through. There is now a vertically placed stitch, embracing a strip of sclera rather wider than the rectus muscle. The first silk stitch (that in the muscle) is now drawn tight and knotted, bringing the cut-end of the tendon forward to its new position. The tendon lies over the complementary suture. Each end of this latter is now passed through the adjoining cut border of the conjunctiva, about 4 mm. from the edge, and the ends are tied firmly, but not very tightly. This stitch brings together the lips of the conjunctival wound and binds the rectus tendon closely to the sclera. The stitches are removed on the seventh or eighth day.

J. B. LAWFORD.

(6) **Van Lint.—Trephining the os unguis and insertion of a rubber drain in chronic dacryocystitis.** *Arch. d'Ophtal.*, Jan., 1920.

(6) **Van Lint** maintains that by a surgical technique much simpler than that of dacryocystorhinostomy a channel between the lacrimal sac and the nasal cavity can be established in cases of chronic lacrimal obstruction. He recommends the procedure to which the above title applies. The operation is easy to perform and the immediate results are "ideal." The newly formed canal, however, is prone to close ultimately, but even then the author thinks the results are at least as good as those of extirpation of the lacrimal sac. A detailed description of the technique is given, the steps of which are, in brief, these: cocain anaesthesia of the region of the sac and of the middle meatus of the nose; plugging of the middle meatus with gauze to avoid injury of the turbinate; the lacrimal sac is exposed and its anterior wall incised in its whole length; the trephine is placed in the lower end of the sac against the bony wall and the lacrimal bone perforated; removal of the plug in the meatus is followed by haemorrhage which is arrested by a strip of gauze pulled through the trephine aperture by forceps; this is followed by a rubber tube, the upper end of which is cut short in the lacrimal sac; the skin wound is sutured over the end of the drain; the nostril is plugged for 24 hours. About the eighth day

physiological serum is injected through the lacrimal punctum; the tubing is removed from the tenth to the fifteenth day. For several weeks the lacrimal passages should be syringed once a week.

J. B. LAWFORD.

BOOK NOTICES

Manual of Ophthalmic Practice. By Col. F. P. MAYNARD, I.M.S. Publishers, Messrs. Thacker, Spink & Co., Calcutta and Simla, India, and Messrs. E. & S. Livingstone, Edinburgh. 1920. Rs. 12.

Col. Maynard's "Manual of Ophthalmic Practice" is obviously the work of an experienced and practical surgeon, but it suffers from the source from which it was derived. This—as stated in the preface—was a series of lectures, in which it is usually necessary, in order to cover the ground in a comparatively short space of time, to deal with many of the subjects somewhat superficially.

The book is lavishly illustrated both with original drawings, and reproductions from other books. It is possibly difficult to obtain the services of an expert medical artist in India, but when so much expense has been devoted to colour reproduction, it is a great pity that the original illustrations in colour are so little illuminating and are not on a more uniform scale. The lack of drawing also in many of them is so glaring that one might infer that microcornea was a common occurrence among the natives of India.

We cannot help regretting that the ripe experience of so capable an ophthalmic surgeon has not been placed before the profession in a more thorough and accurate form.

Manual of Ophthalmic Operations. By Col. F. P. MAYNARD, I.M.S. 2nd Edition. Publishers, Messrs. Thacker, Spink & Co., Calcutta and Simla, India, 1920. Rs. 9. An English Edition is also published by Messrs. E. & S. Livingstone, Edinburgh.

We welcome the second edition of this book; the first edition appeared in 1908, and since that date much new work has been done, particularly in glaucoma. The descriptions of the operations of Elliot, Lagrange, and Herbert are excellent, as are, of course, the chapters on cataract extraction. We note that Colonel Maynard does not advocate the removal of the lens in its capsule.

We think that six pages out of 248 are not sufficient for a full description of the operations on the ocular muscles, but as squint

operations, we understand, are seldom performed in India, this must be the explanation.

The six stereoscopic photographic plates, four on extraction of cataract and two on optical iridectomy and extirpation of the lacrymal sac, are not worth the trouble and expense they must have cost.

Cuban Review of Ophthalmology. (Revista Cubana de Oftalmologia), Vol. II, Parts i and ii, January and June, 1920.

The first fasciculus of the second volume of the Cuban journal, consisting of a massive number of 363 pages with a coloured plate and numerous illustrations in the text, is to hand.

Biographies are supplied of Komoto, E. Rollet, Tomas Blanco, M. Menacho, Louis Dor, Casey Wood, and others; the remainder of the volume supplies original papers and deals with a review of current ophthalmology on conventional lines.

Among the original papers is one by Pacheco Luna, of Guatemala, entitled, "Contribution to the study of Onchocercosis." This paper is illustrated with photographic reproductions of patients who show the filarial tumours; there are also schematic representations of the corneal condition met with in the disease. It is a pity that some of these illustrations have not come out very well. This paper amplifies the preliminary note on this filarial disease, for a description of which see BRITISH JOURNAL OF OPHTHALMOLOGY, Vol. II, p. 536.

The author, with the help of Dr. Rodolfo Robles, has been at work on the life history of the filaria in these cases, and he gives a useful table showing the differences between the Onchocerca Volvulus of Leukhart, 1893, and the filaria of this disease, which he names the Onchocerca sp.

The journal maintains the high standard set in the previous numbers.

CORRESPONDENCE

A SCHOOL OF OPHTHALMOLOGY FOR LONDON

To the Editor of THE BRITISH JOURNAL OF OPHTHALMOLOGY

SIR,—Now that ophthalmology has become a subject for special examinations and diplomas, it is highly desirable that some more comprehensive method of teaching should be carried out than has been done in the past. London, more especially Moorfields, has been the training centre for ophthalmologists all over the world, especially of the English-speaking races. The other ophthalmic hospitals in

London also largely contribute to the teaching of ophthalmology. The teaching of the subjects in these special hospitals is apt to become narrow owing to the fact that the cases which apply for treatment to such an institution are nearly all diseases and abnormalities which are mainly confined to the eye itself, such as errors of refraction and cases requiring operation. Such a training as is received at these hospitals does not give the student a broad enough view of the general bearing of ophthalmology and its relation to medicine. It is extremely desirable that classes for students should be held at other hospitals; as an instance, the nerve hospitals, where more cases of optic neuritis and optic atrophy could be shown in a single afternoon than would be seen in six months at an ophthalmic hospital; further, the causes and treatment of the various nervous diseases which cause ocular manifestations would be far better explained by the neurologist or ophthalmic surgeon who attends that institution than by a member of the staff of an ophthalmic hospital who has not had a neurological experience. All the examinations now rightly require a student to have a sound knowledge of anatomy, physiology of the eye, and optics. Until recently these subjects were not taught at special classes in ophthalmic hospitals, and although now in some hospitals these have been instituted, there are very few ophthalmic surgeons, owing to their lack of early training, who are capable of teaching optics as they should be taught to the student. The provision of lectures in these subjects which are not strictly clinical would be a matter of considerable expense to the medical schools attached to the hospitals. It would be simpler and less expensive for these medical schools to combine to provide apparatus and special lectures on these subjects. Most ophthalmic hospitals in London usually hold one session a day, either morning or afternoon. Students who are going in for examinations and devoting their whole time to work will have nothing to do after the half-day is finished, unless they join more than one hospital and this is usually forbidden to them on the score of expense. It would be extremely desirable that the principal teaching schools attached to the ophthalmic hospitals in London should combine together to draw up a proper course of training for ophthalmic surgeons, and to make arrangements with other special or general hospitals for the students to obtain a knowledge of the diseases associated with ocular manifestations. It would at the same time afford the opportunity to a student of attending and seeing the practice in more than one ophthalmic hospital and provide proper teachers in the preliminary subjects of the examination.

Yours, etc.,

HARLEY STREET, W. M. S. MAYOU.

INTERNATIONAL CONGRESS OF OPHTHALMOLOGY

To the Editor of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

DEAR SIR,—In the November issue of THE BRITISH JOURNAL OF OPHTHALMOLOGY, there appeared an announcement of an International Congress of Ophthalmology, to be held in Washington, in 1922, which is not quite complete, and slightly in error in one respect.

I am authorized by the Committee in charge of the Congress to forward the following notice, with request that you publish it in an early number of the Journal. The notice to read as follows:

Under the auspices of the American Ophthalmological Society, the Ophthalmic Section of the American Medical Association, and the Academy of Ophthalmology and Oto-Laryngology, an International Congress of Ophthalmology will be held in Washington, April 18-22, 1922. The personnel of the temporary organization is as follows:

President	Dr. George E. de Schweinitz
Vice-President	Dr. Edward Jackson
Secretary and Treasurer	Dr. Luther C. Peter
Chairman of Committee on Organization	Dr. Edward C. Ellett
Chairman of Committee on Scientific Program	Dr. Edward Jackson
Chairman of Committee on Finances	Dr. Lee M. Francis
Chairman of Committee on Arrangements	Dr. William H. Wilmer
Chairman of Committee on Membership and Credentials	Dr. Walter R. Parker

Application for membership may be made to the Chairman of the Membership and Credentials Committee, 1025 David Whitney Building, Detroit, Michigan, U.S.A. The membership fee, which includes a copy of the Transactions, is $10.00 in United States money.

If you will kindly insert the above notice in an early number of the Journal, it will be greatly appreciated by the temporary officers of the proposed Congress.

Yours very truly,

WALTER R. PARKER,

Chairman, Membership and Credentials Committee.

December 22, 1920.

SMALL FLAP SCLEROTOMY

To the Editor of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

SIR,—Will you allow me to comment on Colonel Herbert's paper in your issue of December, 1920? I do so with the greatest hesitation on account of my great regard for Colonel Herbert, and of my admiration for the work he has done in the study of glaucoma. As he has named me in this paper, and has obviously referred to me on several occasions therein, I feel that silence on my part might be misconstrued as an admission that I accept the statements made.

He says, "filtration must be through uniformly grey scar lines, *without any trace of fistulous openings*" (the italics are my own). Later, he says, "Some surgeons are still inclined to question the existence of the strictly-filtrating cicatrix, thinking the leakage may be always through fistulous openings, possibly microscopic."

I have always gone much further than this, and have pointed out that his conception of a spongy mass of fibrous tissue allowing fluid to filter through it, is absolutely opposed to everything we know of the behaviour of scar tissue. Such a conception could only be justified, if its sponsor could furnish indisputable anatomical material, demonstrative of the correctness of his theory. Years ago I challenged anyone to bring forward such material, and it has never yet been done. Colonel Herbert illustrates this paper by a section, which he thinks "serves fairly well to illustrate the final condition of the scars which are found clinically to filter permanently and satisfactorily." He says that there was "quite a good area of oedematous conjunctiva but the filtration was insufficient." A few lines further down he adds, "I have little doubt that in this case, the tissue would have ultimately become further organized into a dense impermeable scar." Every pathologist will agree with him in this latter statement; the section is one all too familiar to the glaucoma operator. We know well how in these cases, filtration gradually lessens and the tension rises *pari passu* with the advance of evidence that cicatrisation is taking place in the (sclerectomy) wound. The handwriting is on the wall, and the fact that there is still some filtration under the conjunctiva would deceive no one but a tyro. Colonel Herbert could hardly have produced a specimen better calculated to drive home the lesson with which I have begun this paragraph.

Colonel Herbert gives two reasons in justification of his belief in the filtering as opposed to the fistulous scar: (1) He says, "I do not believe there is ever a leaking hole in a scleral scar which does not give clinical evidence of its existence. Where the leakage is least, there is always a dark point to be seen," etc. He somewhat

modifies this statement in the remarks that follow, but I regret that, possibly due to my own fault, I find it difficult to fathom his exact meaning. Of one thing I can speak with the most absolute certainty: Following a trephine operation, the hole in the sclera may gradually disappear from view; the area of filtration may flatten down, appearing nothing more than an oedematous pad, and yet massage will rapidly increase the local oedema, showing that the fistula is still open. It would be quite impossible for anyone examining one of these cases to say whether a fistula was present or not. I have made enquiries of five other surgeons, with comparatively large experience in trephining, and they all tell me they are familiar with such results. I submit therefore that it is not possible to say from the mere inspection of a scar that it is not fistulous. (2) Colonel Herbert says, " It is no more strange that, bathed in aqueous, the healing process should remain permanently incomplete after these operations, than that it should fail altogether in the centre of a trephine hole. It would be strange indeed if, in not grossly dissimilar cases, there were only the two extremes—complete failure on one hand, and firm union on the other—with no connecting links." If I apprehend clearly the principles at stake, it is just these two extremes that we should expect. We know that if a wound in the iris remains absolutely aseptic, no change whatever takes place in the cut tissues; the line of incision is the same years afterwards as it was when made, there being no evidence of fibrous tissue formation in connection with it. From the first, it seemed reasonable to suppose that the same thing might happen when a hole was made in the sclera as the result of any form of sclerectomy. Experience, backed by anatomical evidence, has abundantly supported this view. Nor is this all, for we can go one step further than this: A mere puncture of the iris in a case of " l'iris bombé," may, we know, remain permanently open, though no tissue has been removed; may we not, on exactly similar lines, explain those cases in which a mere sclerotomy provides a permanent, filtering scar? With these we are familiar, and if we could command them at will, we should have no need of sclerectomy; unfortunately, we know we cannot. If Colonel Herbert had advanced the proposition that, by his method of sclerotomy, a permanent fistula was established in this way owing to the absence of healing in the lips of an aseptic wound, bathed in aqueous fluid, no one could have taken exception to his position; but he does not; he presupposes the formation of a certain amount of fibrous tissue, which remains spongy in nature, and does not go on to ultimate contraction. To me it seems that the presence of this connective tissue in a wound is an indication that that wound is no longer absolutely aseptic, and herein we have to my mind " the gross dissimilarity " which Colonel Herbert denies. Those of us who

have had large experience with any sclerectomy operation, know only too well the appearances which presage the formation of connective tissue in our wounds, and to know them is with all of us to dread them. I hold most strongly that a filtering wound must be absolutely aseptic, or it is doomed to failure. There is no room in these small spaces for half-way conditions.

To me, Colonel Herbert seems to have reversed the scientific method. He has not collected his anatomical facts and founded his theory on them, but he has visualized a conception of a possible condition, and has endeavoured to make his facts fit in with it. I should not write like this if I did not feel a great principle were at stake. One hears men get up at a meeting and say they have gone back to sclerotomy, and that they are satisfied with it. Sclerotomy is as old as de Wecker, and it has been weighed in the balance and found wanting. Lagrange took a big step forward, when he advocated and introduced the operation of sclerectomy, and to advance arguments, however specious, that would draw men away from the sound principle of surgery for which he has contended, is, I hold, to set back the clock of progress. Colonel Herbert, if I understand him rightly, is the antagonist of sclerectomy, and as such I feel bound to oppose him. He asks, "Is it any longer fair to the patient deliberately to aim at the formation of a sclero-corneal fistula, in treating a glaucoma, which can be relieved with certainty by the truly filtering cicatrix of a flap sclerotomy." I submit that he is here begging the question. He has not satisfied the profession that glaucoma *can* be relieved with certainty by the operation he advocates. Nor has he produced any reliable evidence of the existence in fact of what he conceives as "a truly filtering cicatrix."

I have read with bewilderment the following words in his paper: "Take, for instance, trephining as practised by Colonel Elliot. In the milder glaucomas now particularly considered, only a section of the disc is removed, particularly of the deeper layers, and much of the trephine hole is covered by a lid of impermeable corneal tissue. (It does not seem clear that this superficial covering of corneal tissue ever becomes permeable to aqueous," etc.). These statements of Colonel Herbert's on the subject of my work and teaching are in direct opposition to everything I have written from the commencement of my advocacy of trephining. If he will refer to the first edition of "Sclero-corneal Trephining," 1913, p. 56, he will find that I then advocated cutting the disc in such a way as to "leave *a part of its deeper layer* in situ." There is no suggestion that the superficial layers were left. In my second edition of the same work, 1914, p. 68, I advocated cutting off a portion of the trephined disc, "at right angles to the surface, thus leaving the posterior edge of the fistula we aim at making as steep as the anterior edge, which has been cut with the trephine." The same lesson has been pressed

home in my book on "Glaucoma," p. 464. I feel sure that if Colonel Herbert will be kind enough to look up these references, he will acknowledge at once that his statements which I have quoted above are in error, and that they give his readers a very wrong view of my method, and of my clearly-expressed teaching.

There is another statement to the correctness of which I must take exception. Colonel Herbert says, "Attempts to graduate the flow of aqueous in accordance with one's estimate of the needs of individual cases, by altering the size and shape of sclero-corneal openings, seem to have been rewarded with but indifferent success." These remarks are in flat contradiction, not only of my own experience, but of that of many others, who are successfully using my operation. As Colonel Herbert does not use sclerectomy, he cannot be speaking from his own experience. I have read with great care the literature on the subject, and am not aware of any justification therein for his statement.

I have never belittled the danger of late infection, but I think that there has been a tendency on the part of some to exaggerate it, and to lose sight of the fact that it may follow any or all of the modern *successful* operations for glaucoma. I italicise the word "successful," for, as I have so often said, the establishment of a flow of fluid from the interior of the eye to the subconjunctival space, whilst it is the one passport to the cure of glaucoma, brings in its train the possible risk of an infection of the interior of the globe from without. The operations, whose scars are absolutely free of this risk, may be ruled out of the list of reliable procedures; that at least is my estimate of the case.

The really practical point is: how great is this risk of late infection, which we have to set off against the advantages of a successful operation for glaucoma? No one, who has carefully examined the evidence, can fail to be struck with the extraordinary difference in the results of different surgeons. On the one hand, you have Lagrange, Kirkpatrick, Wilmer, and other surgeons, a number of whom have been kind enough to write to me, or to publish their experience—I set aside my own experience for the moment—who have practically no such misfortunes to the debit side of the account of their filtering cicatrices. On the other hand, you have a certain number who have published, and one must honour them for their courage, the most deplorable results. It would be difficult to escape the conclusion—believing, as I firmly do, in the honesty of them all alike—that the personal factor is a very large one, and that many of the unfortunate results recorded might have been avoided by the adoption of a better technique. I know from the letters I have received that not a few of those who believe that they are doing my operation, have never read my writings with any care, and are practising methods of their own

which they believe to be identical with mine, but which are not. I believe that thick flaps, made in accordance with the technique which I have been at much pains to lay down, would, if introduced into the practice of some of these surgeons, revolutionise their results. I would take this opportunity of pleading that they should in future do me this amount of justice.

In conclusion, I wish to comment very shortly on Colonel Herbert's words, "In a few . . . glaucomas . . . I have preferred iris prolapse operation to flap sclerotomy." I am well aware that in different parts of the world, a few surgeons have of recent years advocated iris-inclusion operations, and that amongst these are to be included some very distinguished names. I should be untrue to everything I hold worth contending for, if, while answering other points in this paper, I let this pass unchallenged, much as I would prefer to do so. In our student days we learnt the lesson handed down by men of long and ripe experience, that the inclusion of iris in the lips of a wound was a grave menace, not only to the eye affected, but also to its fellow. As the years have passed over us, and many other lessons have been forgotten or set aside, this one has stood the acid test of time and experience, and we have learnt to dread iris inclusions more and not less than we did in our early days. No array of names, however imposing, and no list of successes, however seductive, can persuade me that it is the right thing to do, to bring about deliberately in our patients' eyes a condition which the whole of our experience goes so strongly to condemn.

Yours truly,
R. H. ELLIOT,
Lt.-Col. I.M.S. Retd.

NOTES

Deaths

WE regret to announce the death of Charles Higgens, consulting ophthalmic surgeon to Guy's Hospital, and ophthalmic surgeon to the French Hospital, London, which occurred suddenly on December 28th, at Pursers, Bramdean, Hants, at the age of 75. The funeral took place on January 1st at West Meon, Hants. A full notice of his career will be published in the next number of the Journal.

Alfred St. Clair Buxton, consulting surgeon to the Western Ophthalmic Hospital, died in a nursing home on December 19th last, aged 67.

Hermann Ulbrich, of Vienna, died in Turkestan on April 1, 1920. His work upon bacteriology and post-operative infections of the eye is well known.

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Honour

THE C.I.E. has been bestowed upon Lieut.-Col. Frederick O'Kinealy, I.M.S.

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Appointments

MR. J. HERBERT PARSONS has been elected President of the Illuminating Engineering Society, in succession to Mr. A. P. Trotter. Mr. Parsons was the first chairman of Council of the Society, has taken a leading part in the sections of its work dealing with the influence of light upon vision, and is a member of various committees on the subject.

Dr. W. C. MacFetridge has been appointed assistant surgeon to the Royal Victoria Eye and Ear Hospital, Dublin.

Dr. A. Gerard East has been appointed ophthalmic surgeon to the Royal Cornwall Infirmary.

Professor Hertel, of Strassburg, has been appointed as successor to Professor Sattler in Leipzig, and Professor Fleischer, of Tübingen, has succeeded to Hofrat Oeller in Erlangen.

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Prize in Ophthalmology

IT is announced that the Lucien Howe Prize of £100 for essays on some branch of surgery, preferably ophthalmology, will be awarded by the Medical Society of the State of New York at the next annual meeting, May 3, 1921. Essays should be in the hands of the chairman, Dr. Albert Vander Veer, 28, Eagle Street, Albany, N.Y., not later than April 1.

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Moorfield's Annual Dinner

IT is now announced that the Annual Dinner of Past and Present Students of the Royal London Ophthalmic Hospital will be held on February 10, 1921, at the Café Royal, Regent Street, at 7 for 7.30 o'clock, and that tickets can be obtained from Mr. Charles Goulden, 42, Welbeck Street, W.1.